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HILUS (AND PERI-HILUS) TUBERCULOSIS AND ITS SEQUELÆ

(ROOT-PHTHISIS)

BY

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By R. MURRAY LESLIE, M.A., B.Sc., M.D.,
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The region of the hilus or root of the lung has long been recognised as a common site of tuberculous deposit, more particularly in connection with the tracheo-bronchial lymphatic glands, but the immense importance of the hilus as an early and primary focus of disease is only just beginning to be appreciated. The frequency with which the lymphatic glands situated close to the bifurcation of the trachea become affected may be readily understood when we recall the fact that these glands are the natural filters to which pass foreign particles, including tubercle bacilli, originally deposited on the bronchial mucous membrane in the act of inhalation or in the alveolar tissue of the lungs, whether originally conveyed there by aspiration or blood infection.

Pulmonary and Extra-pulmonary Lymphatic System.

In order to make it clear how the virus of tuberculosis is carried to the bronchial glands and how the disease itself extends and is disseminated, one must have a right conception of the intra-pulmonary and extra-pulmonary lymphatic drainage system. It has been well said that the whole lung is pervaded in every crevice of its structure by lymphatic tissue. In the alveoli there are branched protoplasmic cells communicating with fine tubes and interstitial spaces lined by endothelial cells, while in the peri-lobular connective tissue there is a fine

* A Lecture delivered at the Medical Graduates' College and Polyclinic on October 8th, 1912.

network of tiny lymph-vessels into which the alveolar lymph passes. In the bronchial mucosa there is a network of lymphatics, the lymph capillaries being situated underneath the vascular capillaries which lie immediately beneath the bronchial epithelium. There is also a network of fine lymph-channels in the bronchial submucosa. Teichmann has shown that the lymphatics of the bronchial mucosa run parallel to the long axis of the bronchi in contra-distinction to those of the submucosa which tend to run transversely on the cartilaginous rings. From the network of bronchial lymphatics lymph-vessels pass to the tissue surrounding the pulmonary blood-vessels accompanying the bronchi. At the point where the bronchi branch we have generally three lymphatic vessels, one of which passes to the artery and the other two to the veins, while the smaller arteries and veins are only accompanied by a single lymph-vessel situated between the blood-vessel and the bronchus. We have thus a series of peri-vascular and peri-bronchial lymphatic channels into which drain the bronchial and peri-lobular lymphatics, and which end in the numerous tracheo-bronchial glands occupying the space below the bifurcation of the trachea and extending on to the pericardium, though single glands are found surrounding the main bronchi and also lying on the pulmonary artery.

Towards the surface of the lung the peri-lobular lymphatics are believed to communicate with the lymphatics of the visceral pleura. These pleural lymphatics form a network of vessels which gradually join up close to the root of the lung into four or five large lymph trunks which terminate in the bronchial glands at the hilus, where there is free anastomosis with the lung lymphatics. The existence of stomata on the surface of the visceral pleura providing direct communication between the pleural sac and the lymphatics of the lung and pleura is doubtful in the light of recent investigations.

It must be remembered also that there is *direct communication between the lymphatic system of the thorax and that of the neck and abdomen.*

As regards the neck, Grober's remarkable experiments demonstrated a direct route to the pleura and lungs *viâ* the cervical lymphatic glands, and the discovery of this route led him to believe that primary infection by way of the tonsils might be the explanation of the frequency with which the apex of the lung was found to be the seat of tuberculous disease. It is certainly fairly common to find that enlargement of the supra-clavicular and axillary glands precedes pulmonary tuberculosis. In the case of tonsillar infection the submaxillary glands are the first to be involved, the disease tending to gradually pass down to the axillary and supra-clavicular glands, and in such cases the bronchial glands are not infrequently found enlarged and caseous. The importance of this direct relationship between the cervical and mediastinal lymphatic systems may be of great importance both in regard to the incidence and spread of pulmonary tuberculosis.

As regards the abdomen, Poirier * proved the existence of free communication between the lymphatics of the pleural and peritoneal surfaces of the diaphragm. As far back as 1903, Professor Behring, of Berlin, maintained that pulmonary tuberculosis was generally the result of infection by the intestinal route, which contention received considerable support some years later from Calmette and Guerin's notable experiments with intra-œsophageal injections of bacillary emulsions in guinea-pigs.

These experiments led these observers to believe that it was the rule for the virus to be carried to the lungs by way of the abdominal lymphatic system instead of the lungs being affected directly by inhalation as had previously been supposed. I understand, however, that both Behring and Calmette have recently considerably modified their views in regard to the respective frequency of infection by inhalation and ingestion, the former being now generally regarded as the more important channel of infection.

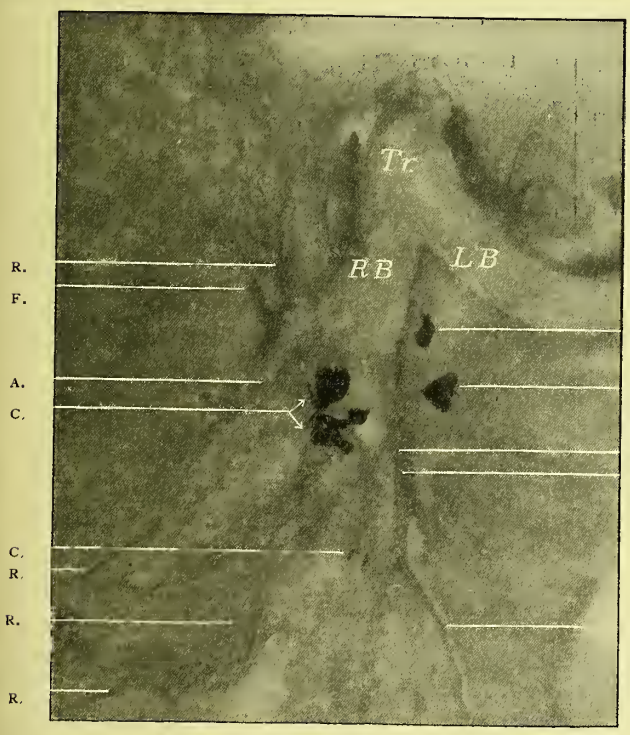
Frequency of Hilus Tuberculosis.

The importance of hilus tuberculosis is shown by the fact that the bronchial lymph-glands are often the first tissues attacked by tuberculosis in early life, and may be the only foci of disease in the whole body. So extraordinarily common are tuberculous foci in the bronchial glands and surrounding lung-tissue that the majority of all persons dying of accident or suicide in Paris, and whose bodies are exhibited at the Morgue, present

* 'Traité d'Anatomie humaine,' Paris, 1902.

evidence of old-standing or recent disease in these parts. Naegeli—the well-known pathologist—found evidence of tuberculosis in no less than 97 per cent. of all adult post-mortem examinations, and in a large proportion of these the disease took the form of calcareous or caseous deposits in the bronchial glands situated either at the roots of the lungs or along the large air-tubes. The remarkable investigations of Hamburger and Monti in Vienna indicated that over 50 per cent. of school-children under six years of age and 94 per cent. of school-children under fourteen years of age had already become infected with tuberculosis, although the natural resistance of the individual was sufficient to prevent 80 per cent. from ever exhibiting symptomatic manifestations of disease. It would probably be correct to state that in the larger number of these children the disease affected the lymphatic glands at the roots of the lungs and elsewhere.

It is, indeed, at the period of infancy and early childhood that the tuberculous seed is commonly sown in the organism, although the disease may remain latent for a prolonged period and only manifest itself when the system is lowered by some acute illness. Professor Nietner, of Berlin, who has specially devoted himself to the study of tuberculosis in childhood, is of opinion that tuberculous deposit often commences in the middle of the second year of life and continues in an



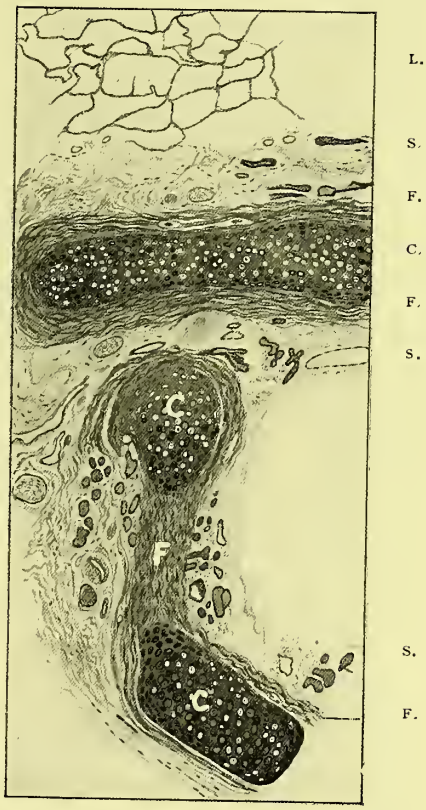


Fig. 1.—Skiagram of healthy lung from post-mortem room. C. Calcareous patches in bronchial glands. A. Air-tubes surrounded by fibrous tissue. F. Thick fibrous sheath round air-tube. R. Radiating shadows due to fibrous tissue; round smaller air-tubes. Tr. Trachea. R.B. Right bronchus. L.B. Left bronchus. (By kind permission of Dr. Jordan.)

Fig. 2.—Microscopical section through same lung shown in Fig. 1 through point s, showing great increase of fibrous tissue around bronchial cartilage (cartilage being transparent to X rays). L. Healthy lung. S. Stroma. C. Bronchial cartilage. F. Fibrous-tissue sheath (tuberculous giant-celled systems sometimes present). (By kind permission of Dr. Jordan.)

increasing ratio every year up to and beyond the age of puberty. Similar observations in regard to the prevalence of tuberculosis in children have also been made by Dr. Phillip, of Edinburgh, who found actual stigmata of pulmonary tuberculosis in no less than one third of the children attending the schools of that city.

The work of Dr. Anton Ghon,* of Prague, on pulmonary tuberculosis in children is one of the most important of recent contributions in connection with glandular and hilus tuberculosis, while in England the radiographic investigations of my colleague, Dr. Alfred Jordan, published under the title "Peri-bronchial Phthisis" have attracted wide-spread attention. Dr. Jordan states that the great majority of a series of "healthy" lungs obtained by him from the post-mortem room and examined by the X rays showed calcareous deposits in the lymphatic glands, either at the roots or along the course of some of the larger air-tubes (Fig. 1).

Fortunately there is a marked tendency to spontaneous cure in the case of glandular tuberculosis, as is evidenced by the frequency with which such calcified bronchial glands are met with post mortem in cases where there had been no suspicion during life of any tuberculous disease. At the same time it must not be forgotten that the disease may lie latent in the glands for long periods, and may be followed many years later by important and serious consequences.

As regards the incidence of the disease, evidence is accumulating that catarrh of the bronchial mucous membrane is one of the most important predisposing causal factors in connection with hilus tuberculosis. Sir William Osler in this way explains the frequent occurrence of tuberculous adenitis of the bronchial glands as a sequela of whooping-cough and measles. Increased vulnerability of the tissues, according to the late Professor Virchow, is the important factor in producing the disease, and such vulnerability would naturally occur as the result of injury or breach of continuity in connection with the bronchial mucosa.

It is also more than probable that when bronchitis has persisted for some time and the ciliated epithelium has thereby become damaged and its functions impaired, bacilli may then find their way by aspiration into the remote bronchial ramifications and may be carried into the alveoli of the lung. Thence they pass between the cells lining the air-vesicles and so into the lymph-channels and connective-tissue spaces of the alveolar walls.

From the frequency with which enlarged bronchial glands and tuberculous thickening in the adjacent pulmonary tissues are found in persons exposed to dust inhalation, it is conceivable that the tubercle bacilli may in many cases reach the bronchial glands by being actually attached to the organic particles, which would thus act as the direct carriers of the infective virus. At the same time the association of silicosis with tuberculosis may also be explained by the theory that inorganic particles—particularly when of a sharp, angular character—may cause sufficient irritation of the glandular and pulmonary tissues to render the resulting inflammatory area a suitable nidus for the growth and development of the bacillus of tuberculosis.

The Primary Site of Chronic Pulmonary Tuberculosis: Apex *v.* Hilus.

Hitherto the apex has always been regarded as the most frequent primary focus of pulmonary tuberculosis. In 1880 Sir James Kingston Fowler* stated that a spot situated from one to one and a half inches below the actual apex was the primary seat of disease in the large majority of cases. This observer also drew attention to the fact that tuberculous disease in its progress through the lungs followed a distinct "line of march" particularly in the chronic and fibroid varieties, the disease spreading in each lobe from above downwards, hardly ever from below upwards. From the primary apical focus he found that the lesion tended to extend in the first instance backwards and then downwards along the anterior portion of the upper lobe in the form of scattered nodules situated about three quarters of an inch within its margin. The apex of the lower lobe became affected at a comparatively early period, the disease tending to spread backwards along the posterior surface of the lung and laterally along the line of the inter-lobar septum. When extension took place along the

* 'Der Primare Lungenherd bei der Tuberculose der Kinder,' Prague, 1912.

* 'Localisation of Lesions in Phthisis,' 1880.

ADDENDUM.

SINCE the publication of this lecture in the *Clinical Journal* the author's attention has been directed to K. Preisich's article in the *Wiener medizinische Wochenschrift*, 1911, Nos. 3 and 5.

K. Preisich, as the result of his investigations on tuberculosis in infancy and childhood, makes the following statement which indicates the importance of peri-bronchial and hilus tuberculosis in children :—

"From the first to the seventh year of life the number of cases of tuberculosis of the lymphatic glands increases year by year. The type is benign probably on account of the great power of resistance possessed by children. . . . After the seventh year tuberculosis of the lungs as a disease, *per se*, attains the greatest importance. . . .

"In children over 8 years of age chronic tuberculosis of the lungs tending to recovery is no longer rare. *Most frequently there are found peri-bronchially disseminated lobular or confluent foci* (probably due to inhalation or infection by way of the circulation). *There is also caseous infiltration starting from the hilus and spreading over an entire lobe* (probably *starting from the hilus glands and spreading back along the lymphatics*).'

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* 'Der Primare Lungenherd bei der Tuberculose der Kinder,' Prague, 1912.

* 'Localisation of Lesions in Phthisis,' 1880.

base it was usually by means of scattered nodules of infiltration, often arranged in a racemose manner. Soon after the apex of the lower lobe was affected there was an involvement of the apex of the upper lobe of the opposite lung, to be followed later by infiltration of the apex of the lower lobe of that lung.

It is important, however, to observe that Kingston Fowler mentions occasional variations in the position of the primary foci and also in the "line of march," and makes special note of so-called cross lesions where the extension seems to take place from the apex of the upper lobe of one lung to the apex of the lower lobe of the other. He also makes special reference to the comparative freedom of the bases of the lungs from tuberculous deposit, stating that primary basic disease is exceedingly rare, there being generally evidence of older lesions of the apices. Fowler attributes the frequency of apical disease to the lesser functional activity of this portion of the lung, which is imperfectly expanded in ordinary quiet breathing, such impairment of function being associated with defective resisting power. It must be admitted, however, that Fowler makes no attempt whatever to explain the rationale of the "line of march" which he so carefully describes. It would seem that a more or less constant "line of march" must have some rational explanation.

On the other hand, Dr. Alfred Jordan* states categorically that chronic pulmonary tuberculosis does not generally commence at the apex, but begins at the roots and extends along the branchings of the large bronchial tubes. I am indebted to the courtesy of Dr. Jordan for permission to reproduce Figs. 1 and 2, already published in the 'Practitioner,' representing calcified glands and peri-bronchial fibrosis.

As regard the mode of infection, and the manner in which the virus gets to the peri-bronchial tissue in the first place, Dr. Jordan does not commit himself to any definite opinion, nor has he given any explanation as to why the peri-bronchial pneumonic process should commence around the large bronchi close to the root of the lung instead of more or less promiscuously around the smaller and more peripheral bronchi.

Dr. Jordan inclines to the view that the primary tuberculous deposit does not take place in the bronchial glands, but in the peri-bronchial tissues in the form of broncho-pneumonic foci, and that the glandular enlargement is secondary.

In a number of cases the infection seems to spread directly to the lungs from the caseous bronchial glands, often apparently by direct contact. Indeed, as Sir William Osler has pointed out, and his statement is confirmed by my own observations, it is sometimes difficult to determine in a section through the affected lung where the caseous bronchial glands terminate and where the pulmonary tissue begins. In the greater number of cases, however, the extension is probably by way of the lymphatic network already described as surrounding the pulmonary vessels and bronchi. It is possible that the extension takes place in a great measure along the peri-vascular lymphatics surrounding the pulmonary vessels, but owing to the close proximity of the pulmonary vessels and bronchi the task of precise discrimination between the peri-vascular and peri-bronchial lymphatics is naturally difficult. It may be that while the larger number of bacilli entering by way of the bronchial mucosa are passed on to the bronchial glands, into which the bronchial, peri-vascular and peri-bronchial lymphatics drain, a certain number of bacilli filter through the mucous, submucous and peri-bronchial lymphatics, and may give rise respectively to tubercles on the surface of the bronchial mucosa, to caseous deposits in the bronchial submucosa, and to broncho-pneumonic areas in the pulmonary tissue surrounding the bronchi. Dr. Jordan favours the view that the tuberculous process generally spreads in the form of broncho-pneumonic extension in the peri-bronchial pulmonary tissues, which in favourable cases may cicatrise into fibrous bands, appearing as dark radiating shadows on X-ray examination of the affected lungs.

Dr. Jordan states that in 40 per cent. of cases of phthisis the disease commences as a definite peri-bronchial infiltration and spreads from the hilus in all directions, but most rapidly along the ascending and descending branches of the main bronchus, and that sooner or later the disease reaches the apex by way of the ascending bronchiole. When the apex is reached, the disease, he says, may there progress rapidly, so

* 'The Practitioner,' February, 1912; 'Brit. Med. Journ., August, 1912.

that the apex appears clinically to be the chief seat of invasion, the primary disease at the root being so deeply placed as to escape detection from without. He explains purely apical cases on the hypothesis that the tuberculous infection has travelled up from the hilus along the main ascending bronchiole to the apex in an early stage, and progresses there, while the track of infection from the root to the apex has healed up, leaving only an excess of fibrous tissue along the ascending bronchiole. Dr. Jordan bases his conclusions mainly on his interpretations of radiographic shadows in the lungs of living subjects, and of similar shadows obtained by the X-ray examinations of lungs taken from the post-mortem room. Now, ingenious and striking as these theories and conclusions are, it is doubtful if Dr. Jordan has so far produced sufficient evidence to warrant definite dogmatic generalisations.

In any case we have here two divergent views—the one ably expounded by Kingston Fowler as the outcome of unrivalled pathological and clinical experience to the effect that pulmonary tuberculosis is primarily an apical disease with gradual extension downwards along a definite "line of march," while the newer view is advanced by an expert radiographer with much less pathological and clinical knowledge, who nevertheless has the courage of his convictions, and boldly states his belief that apical tuberculosis is but an extension of "peribronchial phthisis" which is really the primary manifestation of chronic pulmonary tuberculosis. Is it possible that two views apparently so divergent can be reconciled?

Kingston Fowler* himself, in discussing the pathology of chronic fibroid phthisis, admits that the extension of the disease is more towards the peri-bronchial tissue than towards the alveoli, and states that the bronchi may be thickened from small-celled infiltration, while actual tubercles may be present both in the walls of the bronchi and in the peri-bronchial tissue, in which cases groups of granulations having a racemose arrangement are usually present (see Fig. 3). Fowler also found that the lining membrane of bronchi which pass through such tuberculous broncho-pneumonic areas are often intensely injected and swollen, and may even have undergone extensive tuberculous ulceration. He further found that tuberculous infiltration of the peri-bronchial sheath may lead to marked thickening of the tube and narrowing of its lumen. In some of these cases this fibrous constriction and narrowing of the bronchus may lead to bronchiectatic dilatation beyond the site of constriction.

As far as Dr. Jordan's views are concerned, he does not seem to have fully appreciated, and, indeed, has practically ignored, the frequent occurrence from time to time of autogenous blood infection, aspiration broncho-pneumonia and other accidental contingencies, not to mention the possibility of actual primary alveolar deposit as the result of inhalation. He has disregarded to a great extent the consideration of what the Germans describe as "open" tuberculosis. "Open" pulmonary tuberculosis includes the type in which fragments of muco-pus loaded with autogenous

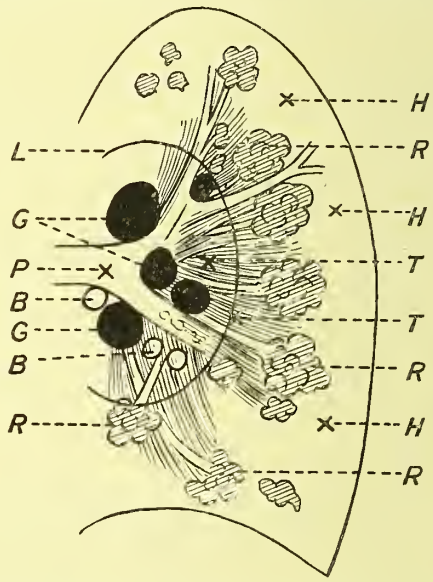

Fig. 3.—Chronic fibroid phthisis commencing as hilus tuberculosis. This drawing was made from a specimen in Brompton Hospital Museum (Catalogue No. 116) and represents a complete section through right lung. Is an excellent example of chronic fibro-caseous phthisis, the disease having evidently commenced at the hilus and extended outwards into the surrounding tissue in a wonderfully symmetrical fashion. P. Pulmonary vessel cut longitudinally. B. Air-tubes cut across. G. Caseous bronchial glands at hilus. T. Tuberculous fibrous thickening and infiltration extending outwards from hilus. R. Racemose tubercle in peri-hilus area. L. Imaginary line circumscribing hilus of lung. H. Peripheral zone of healthy lung-tissue ¾ in. wide. (Drawing by author.)

* 'Diseases of the Lungs,' Fowler and Godlee.

bacilli may pass directly into the lumen of the bronchi, thus forming what might be described as tuberculous "bronchial emboli," capable of being aspirated into the terminal bronchioles and alveoli as well as of being expectorated in the sputum. Dr. Jordan's views regarding the universality of peri-bronchial extension might be quite sound if he were dealing only with a "closed" disease with no bacilli in the bronchial contents and spreading along the lymphatic lines of least resistance, but there is in actual fact a constant liability to accidental contingencies. On the one hand we may have the erosion of a blood-vessel by a tuberculous gland or other diseased focus, so that the virus is carried by the blood-stream and deposited at the less resistant apices or elsewhere, while on the other hand we have the above-mentioned tuberculous "bronchial emboli" which may be carried by inspiratory efforts to the apices and peripheral portions of the lungs possibly at a comparatively early stage of the disease, just as occasionally happens in the case of the classical "phthisis ab hæmoptoe" when new deposits occur after a pulmonary hæmorrhage due to the aspiration of the infected blood into distant portions of the pulmonary tissue.

The presence of autogenous bacilli at an early stage in the bronchial secretions may indicate one of several processes. There may be small miliary tubercles in the bronchial mucous membrane, which, on breaking down, form small tuberculous erosions discharging tubercle bacilli into the bronchi; there may also be present large caseous nodules in the submucous tissue which may burst into the lumen of the tube; and more important still in the course of the tuberculous broncho-pneumonic extension above referred to, it is quite common to find in addition to and *pari passu* with the cicatrising fibrous process already described, that some of the broncho-pneumonic patches may caseate, not a few breaking down into minute cavities communicating more or less freely with the adjacent bronchioles, into which the tuberculous contents of the alveoli escape.

In any or all of these contingencies which are so common in the later stages of the disease to as practically constitute an essential part of the pulmonary tuberculous process, we have all the conditions necessary to bring about an aspiration broncho-pneumonia. Fragments of muco-pus loaded with bacilli may be sucked in with inspiration and carried to the apices and other portions of the lungs, where they may lodge and set up tuberculous inflammatory mischief. The ciliated epithelium is no longer able to expel such fragments, and the phagocytic action of the lining cells of the bronchi, bronchioles and alveoli, though generally quite effective in the case of the few individual air-borne bacilli carried from without by inhalation, may be no longer capable of dealing with such concentrated masses of bacilli, accompanied as they are by degenerated epithelial *débris*, which may itself act as an irritant in addition to furnishing pabulum for the accompanying bacilli, until they succeed in obtaining a fresh nidus in the pulmonary tissue suitable for their growth and development. Areas of tuberculous broncho-pneumonia are thus formed as independent foci, and these are specially apt to occur at the apices of the lobes, where there is least functional activity and consequently least power of resistance.

The frequency of the pulmonary apex as the seat of tuberculous disease may be explained on the supposition that its normal inactive condition in the course of normal respiration induces a certain degree of local anæmia (ischæmia). The result is that we have the very conditions most likely to favour the deposit and development of the tubercle bacillus, such deposit not being surrounded by well-filled blood-capillaries which are uniformly present in the actively functioning areas of the lung at the base and elsewhere. At the apex the deposit is inadequately bathed by blood-serum containing protective and anti-bactericidal properties; there is, in short, as Sir Almroth Wright would express it, "defective lymph lavage," thus converting the apical focus into a "non-bacteriotropic nidus."

The advocates of the view that the hilus is generally the primary focus of pulmonary disease find it difficult to determine whether apical infiltration occurs independently of *Hilus tuberculosis* as patients seldom die at the early stage, unless from some extraneous cause. In the case of patients who die with advanced disease of one lung, and are found post mortem to have early apical deposit in the opposite lung, there are always hilus deposits, and therefore such cases throw but little light on the problem.

The principal evidence in support of the hilus as

a primary focus of disease is obtained from radiographic examinations of early cases, and this evidence is by no means absolutely conclusive. The difficulty arises from the fact that in the lungs of nearly all presumably healthy persons we find opaque areas at the roots situated just outside the cardiac shadow, while linear radiating shadows —the so-called "hilus shadows"—pass out into the lung substance for varying distances. There is at present considerable difference of opinion in regard to the significance of these hilus shadows. Some of the German radiographers, indeed, regard these radiating shadows as representing the normal pulmonary vessels filled with blood, while other radiographers are equally positive that they are definitely pathological. Thus Dr. Jordan states that they are due, in the majority of cases, to the previous entry of tubercle bacilli and to the fibrosis resulting from the reaction of the organism to render these bacilli inert.

Hilus shadows and blotches and radiating striations of varying density confined to the roots are practically universal, and invariably present to a greater or less extent in all adults. Accordingly experienced radiographers, such as Dr. Ironside Bruce and Dr. Jordan, are of opinion that *per se* they have but little significance in adults except as being indicative of old inflammatory deposit—tuberculous or otherwise.

The fact that Naegeli and other pathologists found evidence of tuberculous deposit almost constantly at the roots of all lungs examined by them post mortem certainly lends support to such a theory, though it does not actually prove the correctness of the interpretation of the X-ray hilus shadows. In any case, as long as the shadows are confined to the root area, it is at present extremely difficult to differentiate between old-standing probably cured disease contracted in childhood and recent tuberculous infiltration. In the majority of adult cases exhibiting shadows strictly confined to the roots it is safe to conclude that if they are pathological they indicate merely old-standing cured disease contracted in childhood and not recent tuberculous infiltration.

Another difficulty arises from the fact that in long-standing bronchitis, bronchiectasis and silicosis one finds similar, though possibly coarser, radiating shadows, which may be the result of cicatrised inflammatory areas due to non-tuberculous irritation (Fig. 10). Here, again, however, it is difficult to exclude tuberculous infiltration as a secondary process, which is undoubtedly a common accompaniment in gold-miners' and tin-miners' phthisis and other forms of pneumokoniosis. If in doubt it is often well, in the absence of tubercle bacilli in the sputum, to have recourse to the subcutaneous tuberculin test. Well-marked hilus shadows, particularly if they extend into the lung substance well outside the root area in conjunction with a positive tuberculin test, I believe to be extremely significant of tuberculous disease.

There are, however, other important X-ray appearances which are of great confirmatory value. It has long been known that restricted movement of the diaphragm, as seen through the fluorescent screen, is frequently present in early pulmonary tuberculosis, such restricted movement occurring before any definite shadow at the apex or other portion of the lung tissue proper becomes evident. May it not be that this restriction of movement may, in reality, be due to hilus disease (glandular, or pulmonary, or both) restricting the air entry?

A vertically placed tube-like heart is another confirmatory sign, and is supposed to be characteristic of tuberculous soil and to be one of the dystrophies which predisposes to tuberculosis. When the heart occupies its normal oblique position the root opacities are not of such importance.

Any X-ray evidence of tuberculous deposit outside the root area is of course highly significant. One might specially mention the imperfect lighting-up with inspiration of individual areas of the lung-tissue on examination with the fluorescent screen; also grape-like clusters of minute opacities occurring outside the root area which generally indicate foci of tuberculous broncho-pneumonia (see Fig. 4).

It is difficult to draw a hard and fast line of demarcation between the hilus proper and the pulmonary tissue outside the hilus—the peri-hilus. When viewed antero-posteriorly a vertical line drawn through the costo-chondral articulations affords a fairly accurate dividing line for ordinary clinical purposes (see Figs. 4, 6, 9, 10).

Incidentally it may be mentioned that in taking a radiograph of the lungs it is most important that the patient should hold his breath during the time of exposure to the X-rays.

In examining for enlarged glands in the region

of the hilus a lateral oblique examination should always be made, as this frequently reveals a considerable enlargement of these glands, which otherwise would escape detection as they are often concealed by the normal median shadow when examined antero-posteriorly. If much enlarged, they may cause a marked increase of the normal median opaque area (produced by the sternum, aorta and upper dorsal spine), and this broadening of the mediastinal shadow may be so pronounced as to simulate aortic aneurysm. The presence of very dark root opacities indicating calcified foci definitely proves the existence of tubercle in a quiescent or cured condition, and is strong evidence that the

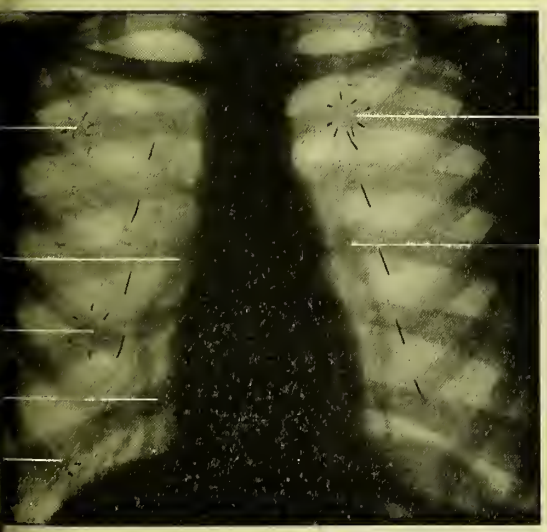

Fig. 4.—Skiagram of chest from supposed case of spasmodic asthma following whooping-cough in boy, æt. 9 years—in reality a case of hilus tuberculosis. Note blotching (H) at roots of both lungs (most distinct on right side) lying external and parallel to the narrow vertically placed cardiac shadow. At the right hilus there are two small opaque spots suggesting old calcified foci (C). Linear shadows may be traced passing towards the periphery and extending in the direction of the apices as high as the level of the second rib in front. Here and there throughout both lungs may be seen small rounded opacities (F) arranged in clusters, indicating foci of tuberculous broncho-pneumonia. *N.B.* — After three months' open-air treatment at Bournemouth patient gained one stone in weight, and the asthmatic attacks entirely disappeared. (Radiogram by Dr. Ironside Bruce.)

hilus root shadows are probably also tuberculous in origin (see Fig. 5).

It goes without saying that the well-known mottling and dark shadows so characteristic of active tuberculous disease in a more advanced

Fig. 5.—Skiagram of chest of a boy, æt. 11 years, admitted to Royal Chest Hospital with obscure symptoms suggestive of pulmonary tuberculosis, but no definite physical signs. There are numerous calcified foci in the hilus and peri-hilus areas on the right side, apparently situated along the ramifications of the air-tubes and pulmonary vessels. Linear shadows may be seen on both sides passing out from the root blotches in the direction of the right apex and left base. (Radiogram by Dr. Jordan.)

Fig. 6.—Skiagram of hilus tuberculosis (supposed bronchial catarrh). From case of gentleman, æt. 24 years, with three months' history of obstinate cough and slight loss of weight, but no physical signs except deficient expansion of chest. Note the well-marked hilus blotches, some of which may represent calcified glands, and also the linear striations passing upwards and outwards from the root area. There was defective movement of diaphragm. The hilus shadows indicate old healed tuberculous deposit, while the small rounded opacities observable here and there are probably due to more recent tuberculous foci. (Radiogram by Dr. Ironside Bruce.)

stage would be even still more confirmatory, but here we are dealing mainly with pulmonary tuberculosis in its earlier manifestations, before the various accidental contingencies of autogenous infection and aspiration broncho-pneumonia have occurred.

Apart, however, from X-ray evidence, there is a considerable and growing amount of pathological evidence in support of hilus tuberculosis as an early manifestation of pulmonary disease. It is quite common to meet post-mortem with cases of fairly

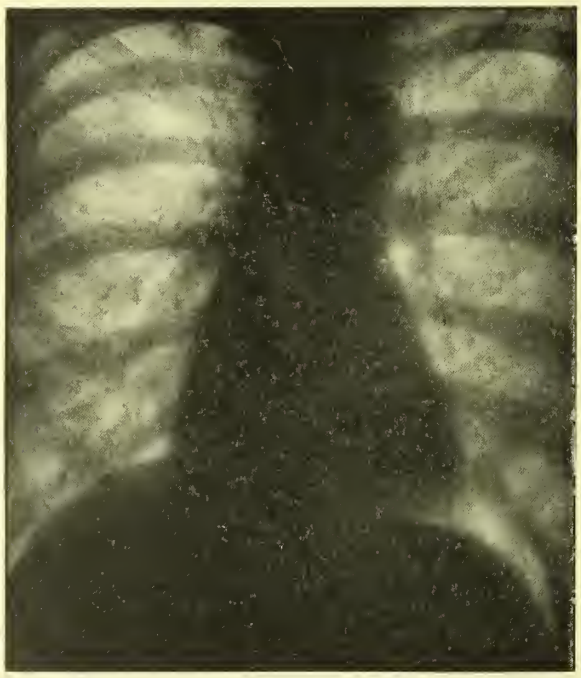

Fig. 7.—Skiagram from lady, æt. 44 years, with history of recurrent hæmoptysis, but no cough and no definite physical signs, except deficient air-entry at right apex. Well-marked reaction with 5 mgrm. of old tuberculin. Appearances indicate old hilus tuberculosis. Note the hilus blotches with coarse linear shadows passing towards apices and right base, indicating fibrous thickening along the air-tubes. The dark rounded root opacities probably represent enlarged bronchial glands. (Radiogram by Dr. R. Knox.)

extensive disease more or less limited to the root area and to the lung tissue immediately surrounding the root area, while the apices and peripheral portions of the lungs may be wholly or comparatively free from tuberculous infiltration. Fig. 3 is a good illustration of this type of case, of which I have seen a considerable number, in which extensive central disease is surrounded by a peripheral shell of healthy pulmonary tissue.

I have already referred at length to the almost universal presence in all post-mortem examinations of tuberculous deposit in the tracheo-bronchial glands and surrounding pulmonary tissue in the neighbourhood of the hilus.

The Retro-Impulsion of Lymph as an Explanation of Hilus Tuberculosis.

The *retro-impulsion of lymph* is now a well-proved phenomenon, the back-flow taking place

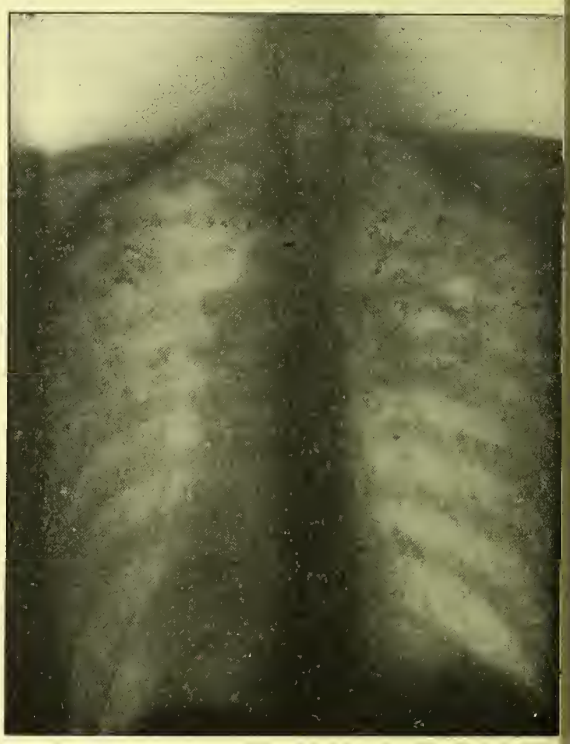

Fig. 8.—Skiagram of chest of young lady, æt. 32 years, showing spread of tuberculosis from hilus to apices, particularly on the right side, where the mottled areas may be seen filling up the space between the hilus shadow and the apex. Note also the elongated tube-like heart. Notwithstanding the history of a slight cough for two years, and the presence of tubercle bacilli in the sputum and the X-ray evidence of extensive disease of the right upper lobe, patient looked wonderfully well, and the physical signs were mainly confined to the right inter-scapular region, where there was slight dulness, prolonged expiration, and bronchophony. (Radiogram by Dr. Alfred Jordan.)

along the distended lymph-channels, the valves of which are no longer competent. Sir Almroth Wright has observed this backward flow of lymph as being of frequent occurrence in bubonic plague —the extension taking place against the usual

course of the lymph-stream. The same phenomenon may be observed in the case of tuberculous cervical glands in childhood, the extension of the disease occurring in the opposite direction to the lymph flow. Experimentally it is comparatively easy to inject such a substance as adrenalin into the subcutaneous tissue of a limb and make it pass from the proximal to the distal portion of the lymph channel. This, indeed, is the principle of ~~Bier's~~ treatment of distal disease by a proximal bandage of a limb, and is also the rationale of Sir Almroth Wright's back-massage in the treatment and cure by lymph lavage of tuberculous and inflammatory conditions of the terminal portions of the extremities.

Let us now apply the doctrine of retro-impulsion of lymph to explain the incidence of hilus tuberculosis.

That there is naturally a special predisposition to tuberculous deposit in the region of the hilus of the lung will appear from the following considerations :

(1) The great proportion of the foreign particles in the inspired air, including tubercle bacilli, impinge and lodge on the mucous surface of the trachea and larger bronchi. The bacilli, once they come in contact with the moist bronchial mucosa, are thereby imprisoned and their further progress along the bronchial tree is arrested. Many of these are killed by the phagocytes, while a few may escape. These are absorbed by the pigment-cells of the mucosa, or find their way through some breach of continuity and pass on by way of the bronchial and peri-bronchial network of lymphatics towards the hilus in the direction of the lymph-stream.

Further, all the living bacilli which have escaped the phagocytic action of the cells of the bronchial mucosa and connective-tissue stroma, in addition to those which may have been derived from the cervical and abdominal lymphatic systems, become eventually concentrated in the region of the root, containing, as it does, the terminal portions of the lymphatic drainage system of the thorax.

(2) After a time—it may be a period of years lasting from infancy to puberty—the bronchial lymph-glands which are the ultimate destination of the bacilli and other foreign particles may become caseous and eventually calcareous, so that the lymph-stream is blocked and the afferent lymph-vessel walls become distended, particularly at the terminal portions, so that the valves are no longer competent and a slow backward flow of lymph sets in.

(3) As a result the tubercle bacilli present in the sluggish backward-flowing lymph-stream filter through the walls of the distended lymph-channels and set up a broncho-pneumonic process in the surrounding pulmonary tissue. As these inflammatory areas heal up and cicatrise, the terminal lymph-vessels become still further obstructed by the cicatricial tissue, and so the filtering, inflammatory, and healing process is repeated a little further out, the extension taking place slowly and steadily from the root towards the periphery and mainly in the tissues surrounding the bronchi and pulmonary vessels.

(4) The whole inhalation process is as a general rule so extremely slow (in the absence of accidental contingencies producing autogenous infection) that there is as a rule plenty of time for protective antibodies to be formed in the system and for immunity to be gradually established. In this way the hilus tuberculous infiltration gradually entirely heals and cicatrises, while the increased phagocytic efficiency of the bronchial mucosa prevents the access of fresh living bacilli. This affords a rational explanation of the fact that although according to Hamburger and Monti 94 per cent. of the population under fourteen years of age have actually been infected with tuberculosis, fully 80 per cent. of these never exhibit symptoms or signs of manifest disease.

The retro-impulsion of lymph theory, of course, presupposes the hypothesis that the glands are the primary seats of tuberculous deposit, which is no doubt true in a large proportion of cases. It is probably equally true that in a certain number of instances broncho-pneumonic extension in the peri-bronchial and peri-vascular pulmonary tissue is a primary phenomenon and the result of direct infection from the bronchial mucous membrane with secondary involvement of the glands, the extension in such cases taking place in the direction of the lymph flow, the glands acting as the natural barriers in preventing systemic infection.

Confirmatory evidence of the importance of tuberculosis spreading from the hilus into the lung-tissue is obtained from a study of the various forms of pneumonokonioses, such as anthracosis, silicosis, etc. In the case of anthracosis (coalminers

disease) the carbon particles are found deposited mainly in the bronchial glands, while fibroid changes usually begin in the peri-bronchial lymph-tissue, and, indeed, in the early stage of anthracosis the fibrotic changes are almost entirely confined to these parts. We get limited patches of fibroid broncho-pneumonia, which here and there may coalesce into greyish-black areas of fibrosis. Cornet states that similar conditions govern the inhalation of tubercle bacilli and that the greater number of the inspired bacilli are deposited on the ciliated epithelium of the upper passages and tend to be swept outwards by the current of mucus, though some find their way through the mucous membrane, and are carried by the lymph-stream to the bronchial glands, which occurs with special frequency in the case of children.

The rapidity of the extension of the tuberculous process in the lungs is as a rule inversely proportional to the degree of immunity—natural or acquired—the determining factors being not so much the numbers or virulence of the invading bacilli, as the resistive power of the invaded tissues on the one hand, and the degree of vulnerability and phagocytic efficiency of the bronchial mucous membrane on the other. This, indeed, is the probable explanation of the difficulty of producing tuberculosis of the lungs in guinea-pigs by the experimental introduction of bacillary emulsions into the trachea.

The fact that most of us have had at one time tuberculous deposit now represented by some fibrous thickening at the roots may in reality be an advantage. A definite degree of immunity has thus been established, and we are actually in a better condition to resist any future invasion than if we had never contracted the disease at all. It is probable, indeed, that the gradual reduction in tuberculosis mortality is largely due to the fact that the population is gradually becoming immunised in this way. This theory will serve to explain the alarming spread of, and mortality from, tuberculosis among the native populations of Bengal, the West Indies, Australia, and elsewhere, who have not been immunised.

The resistive power of the tissues depends mainly upon heredity, but also upon dietetic and other environmental conditions, while the vulnerability of the bronchial mucous membrane, as already mentioned, is greatly increased by catarrhal conditions such as are so frequently met with as accompaniments of measles and whooping-cough, which necessarily impair its integrity.

Sequelæ of Hilus Tuberculosis.

(1) *Chronic fibroid phthisis.*—Though there is in the large majority of cases of hilus tuberculosis a tendency to a natural cure of the malady, it is far otherwise in the minority of instances which ultimately exhibit symptoms and physical signs of manifest tuberculous disease. In a certain proportion of cases, probably as the result of individual lack of resistance, hereditary or acquired, the broncho-pneumonic areas, instead of healing up and cicatrising, continue to spread and coalesce with other similar foci to form larger patches of racemose tuberculous broncho-pneumonia. These, though undergoing a certain amount of fibrotic transformation, slowly but steadily advance until eventually there is produced the familiar condition known as chronic fibroid phthisis, which in some cases may reach a fairly advanced stage before it can be detected by the ordinary clinical methods of physical examination owing to the fact that the disease may not have reached the peripheral portions of the lung, which are alone accessible to the clinical observer through the medium of palpation, percussion, or auscultation.

Fig. 3 is a drawing of a specimen in the Brompton Hospital Museum from the case of a man, æt. 34 years, who died in the hospital. The following is the description of the naked-eye characters of the right lung taken from the Museum Catalogue published in 1904: "Numerous recent racemose tubercles with pigmented centres and caseating circumferences are seen *affecting the central portions of the upper and lower lobes, a shell of practically unaffected lung-tissue about two thirds of an inch thick separating them from the pleural surface.* This specimen explains how in some cases extensive tuberculous disease of the lungs may be present and yet the physical signs may be scanty or altogether absent."

In many cases, however, long before this advanced stage is reached, there is physical evidence of disease at the apex or elsewhere (Figs. 7, 8, and 9).

Whether it is true, as Dr. Jordan maintains, that the tuberculous infection often travels in the

peri-bronchial tissue from the hilus to the apex at an early stage, the disease progressing at the apex while the track of infection from the root heals by a process of fibrosis, has not yet been confirmed by other observers. I think this apical complication is quite as likely, at any rate in a considerable portion of cases, to be due to some accidental contingency producing autogenous infection, whether by way of the blood-stream or bronchial channels, or possibly even by direct exogenous infection through inhalation.

The view that apical tuberculosis is *always* secondary to hilus tuberculosis spreading upwards by means of peri-bronchial extension cannot be maintained, but that such a sequence occasionally occurs at a comparatively early stage is more than probable.

In Fig. 3 the disease seems to have extended more or less continuously in all directions, though the principal extensions are along the pulmonary vessels and air-tubes.

(2) *Tuberculous pleurisy.*—The great majority of subacute and chronic tuberculous pleurisies, whether dry or accompanied by sero-fibrinous effusion, are tuberculous in origin, although there may be nothing in the lungs or elsewhere to suggest the presence of tuberculosis. Sir William Osler states that in almost every instance of tuberculous pleurisy there are really tuberculous foci in the bronchial glands or lung-tissue. I have already mentioned that the pleural lymphatics join up into four or five large lymph-trunks, which terminate in the bronchial glands at the hilus. Applying here also the doctrine of the retro-impulsion of lymph, the ready extension of the tuberculous process from the hilus to the pleura is obvious. The extensive tuberculosis of the pleuræ and the mediastinal glands so frequently found in cattle is significant in this connection.

I have submitted many patients with tuberculous pleurisy to X-ray examination, and in most cases there was evidence of hilus infiltration.

(3) *Asthma.*—In a large proportion of cases asthma *in children* is really due to reflex or direct irritation from enlarged bronchial glands and peri-bronchial thickening and infiltration. I have lately had several children suffering from asthmatic attacks examined by X rays, and almost invariably we have found unusually well-marked hilus shadows, and not infrequently dark discrete areas indicating calcified glands. Fig. 4 is an excellent illustration of such a case. In some cases large glandular masses have been discovered, particularly when viewed from the right and left lateral aspects. Occasionally the thoracic nerves may become involved in the hilus thickening, particularly the recurrent laryngeal branch of the vagus nerve, and such nerve-compression may have an important influence in the production of the asthmatic attacks, although in children of a nervous diathesis the mere irritation and compression of the bronchial tubes may of itself be sufficient to induce asthma. In adults also asthmatic attacks are undoubtedly in some instances associated with hilus tuberculous thickening (Fig. 9).

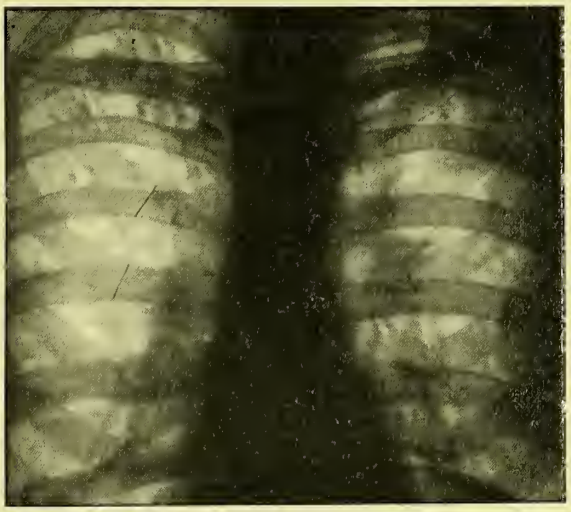

FIG. 9.—Skiagram from case of chronic fibro-caseous tuberculosis (supposed asthma). Case of gentleman, æt. 39 years, with twelve months' history of asthmatic attacks. Had occasional cough and expectoration, which contained tubercle bacilli. The physical signs consisted of a few crepitations at the right anterior apex, numerous rhonchi scattered over both lungs, and impaired expansion of both sides of the chest. Note the elongated cardiac opacity, the extensive hilus blotches on both sides, the well-marked striation and numerous rounded opacities in both lungs, and particularly the finger-like processes extending from the hilus to the right apex. (Radiogram by Dr. Ironside Bruce.)

(4) *Acute miliary tuberculosis.*—Evidence is accumulating in support of C. Weigert's hypothesis that miliary tuberculosis, whether pulmonary or general, is not only due to a blood-infection, but is often associated with tuberculosis of blood-vessels, and probably the most important sites of such vascular tuberculosis are the branches of the pulmonary veins. Caseous bronchial glands not infrequently become adherent to the adjoining pulmonary veins, penetration of which may lead to systemic infection. In rare instances a large caseous glandular or other focus may actually erode the vessel, thus discharging into the blood-stream an enormous quantity, not only of bacilli, but of their toxins also; such a phenomenon would readily explain the occasional extensive

diffusion of the infection, involving in some instances the tissues of the whole body (including the peritoneum and meninges), and would also account for the high pyrexia due to profound toxæmia.

There is also the possibility at any time of a large number of bacilli being discharged into the thoracic duct or right lymphatic duct, and passing by way of the large veins at the root of the neck into the right side of the heart and so into the pulmonary circulation.

Diagnosis.

While the symptoms of quiescent hilus tuberculosis are practically *nil*, those of active hilus tuberculosis are by no means characteristic and are almost identical with those of slight bronchial catarrh, being differentiated only by their persistence and a more marked degree of failure of health. Certain symptoms are suggestive—obstinate dry cough, dyspnœa, slight hæmoptysis, feeling of tightness in the chest, wheezing (? asthmatic) attacks (particularly in children), occasional evening rises of temperature and increased frequency of pulse in conjunction with a varying degree of anorexia, languor, anæmia, debility and loss of weight.

As regards hæmoptysis, it is not uncommon to meet with patients in whom a pulmonary hæmorrhage has been the one and only symptom suggestive of pulmonary tuberculosis. In not a few of these cases, particularly when the patient has been placed under suitable hygienic conditions, no symptoms ever develop and doubts are often expressed as to whether the patient had tuberculosis at all. I believe that many of these cases belong to the category of central tuberculosis starting in the root area, in which a caseating focus has accidentally ulcerated into a small blood-vessel.

As regards physical signs, the disease is so deeply situated that it may reach a fairly advanced stage before giving any external indication of its presence, as it is only the superficial portions of the lung that are really accessible to the ordinary methods of clinical investigation. In this respect hilus tuberculosis resembles central pneumonia, and resembles it still further in the fact that the occurrence of a few crepitations at any one point often indicates, not a small superficial lesion, but an extensive deep-seated one, which at this particular point has reached the surface of the lung. The hearing of a few such crepitations in the infra-clavicular region or supra-spinous fossa is apt to mislead the physician and induce him to diagnose early apical tuberculosis when in reality he may be dealing with advanced central disease (see Fig. 3) with an apical extension. Just as an experienced dentist will diagnose extensive dental caries from the observation of a single black speck situated on the surface of a supposed healthy tooth, so in like manner a careful physician will often suspect extensive hilus and peri-hilus tuberculosis from the presence of apical signs in association with impaired movement—a suspicion which can be readily confirmed or negatived by a modern X-ray examination.

In a certain proportion of cases I have obtained important evidence from a careful examination of *the interscapular region.* It is not uncommon to find close to the root of the spine of the scapula impaired percussion, harsh bronchial breathing, prolonged expiration and increased voice-conduction (bronchophony and pectoriloquy (see Fig. 8). In some instances there is deficient air-entry at one base, while basal rhonchi, for the most part unilateral, are by no means infrequent. My experience leads me to believe that the presence of *persistent rhonchi in a suspiciously tuberculous case often indicates central tuberculosis spreading from the hilus.*

Deaths from bronchitis in middle-aged persons are rightly attributed by insurance offices to pulmonary tuberculosis.

In cases with recent tuberculous deposit there is generally a well-marked tuberculin reaction, which confirms the diagnosis by indicating the presence of some active focus of disease.

In all instances where deep-seated disease is suspected recourse should be had to *radiography*, which *is by far the most important and often the only method of diagnosing hilus and peri-hilus tuberculosis.* Indeed it is practically impossible to form any adequate opinion of the extent of central disease apart from X-ray examination. To my mind, one of the greatest services rendered by radiography consists in the valuable information it affords in regard to morbid thoracic conditions.

It is indeed quite remarkable what advanced broncho-pneumonic and fibroid changes may be revealed by the fluorescent screen and radiogram, quite unsuspected even after a most careful physical examination, in cases which were presumed either to be quite in the incipient stage or free from active disease altogether. This is particularly the case when the condition is complicated by emphysema of the lung. The hyper-resonant percussion-note masks any underlying dulness, while the accompanying bronchitic signs (whether rhonchi or *râles*) make it impossible to differentiate the adventitious sounds characteristic of tuberculous infiltration, although the concentration of bronchitic signs in the upper parts of the lungs may make one suspicious of serious underlying disease. Fortunately these cases, so difficult to diagnose apart from the presence of tubercle bacilli in the sputum, are readily diagnosed by X-ray examination, as the clear emphysematous lung-tissue stands out in marked contrast to the deep mottlings, blotches and shadows of tuberculous disease (see Figs. 9 and 10).

I have already discussed the characteristic X-ray appearances, including hilus blotches and linear shadows indicating fibrous thickening at the root, and the mottlings and clustered opacities of active tuberculous broncho-pneumonia.

Blotches, opacities and linear shadows at the actual root are so common as to be considered almost normal—"the normal hilus shadows." On the other hand, shadows outside the hilus proper are much more significant, and definitely mottled or stippled areas in the peri-hilus tissue, particularly if they assume a clustered or racemose form, are highly suggestive of active tuberculous disease.

I have referred to the difficulty of diagnosing tuberculous infiltration at the roots from silicosis, and other forms of pneumokoniosis, and from non-tuberculous chronic inflammatory deposits.

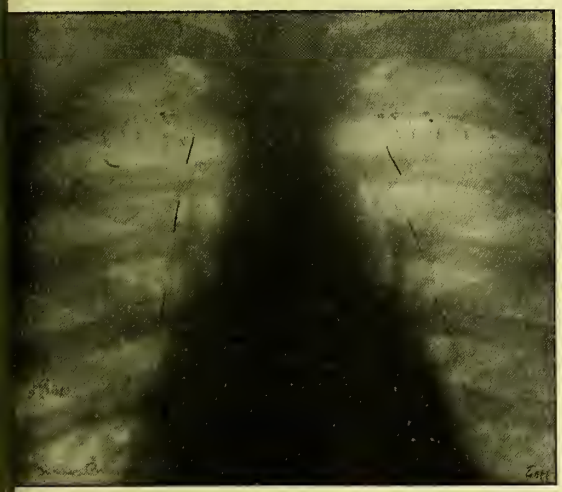

Fig. 10.—Skiagram of case of silicosis, from gentleman, æt. 40 years, who had been exposed for many years to inhalation of silicious dust in South African gold mines. Note the increased opacity of both lungs, more particularly in the region of the hilus, with well-marked linear shadows extending outwards into the emphysematous lung tissue. Was for some time troubled with spasmodic cough. After two years in fresh air all symptoms disappeared, and patient is now quite well. (Skiagram by Dr. Ironside Bruce.)

My experience leads me to confidently state that it should be made a definite rule for all children and young adults who manifest the symptoms of persistent bronchial catarrh, particularly if accompanied by anæmia, lassitude, or other signs of failing health, to be submitted without delay to an X-ray examination.

Prognosis.

The earliest stage of hilus tuberculosis is one of the most curable of all morbid conditions, as is proved by the fact that the great majority of all lungs exhibit post-mortem evidence of healed tuberculosis in the root area, whether in the form of calcareous deposit or fibrous thickening. In the case of hilus tuberculosis not strictly confined to the root area, but extending into the immediately surrounding pulmonary tissue—the peri-hilus—the prognosis is as a rule extremely favourable if the patient's environment is a suitable one. When, however, the disease has reached the apex or other superficial portion of the lung giving rise to physical signs, the outlook is much less hopeful.

Generally speaking a good prognosis depends mainly on two factors:

(*a*) *The degree of resistance of the individual.*

(*b*) *The early diagnosis of the morbid leison.*

(*a*) The degree of resistance depends largely on the comparative immunity and phagocytic efficiency derived from a good heredity, and also to a great extent on favourable environmental conditions. The ubiquitous tubercle bacillus probably obtains lodgment in the pulmonary root area of each and all of us sooner or later, and yet in over 80 per cent. of us the natural resistive power of our tissues is sufficient to effect a permanent cure. This proves the paramount importance of the natural resistive forces of the normal healthy individual. That environment as well as heredity plays an important part is proved conclusively by the fact that tuberculosis claims its victims mainly from the poorly fed, poorly clothed and poorly housed members of the community.

The importance of environment is still further borne out by a comparison between hospital and private patients suffering from tuberculosis. Thus in Dr. Pollock's Brompton Hospital statistics the average duration of life was two and a half years, as compared with Dr. Theodore Williams's statistics taken from the records of a thousand consecutive private patients, whose average duration of life was seven years.

(*b*) As regards the *value of early diagnosis* it is impossible to over-estimate its importance. The late Dr. Gee was of opinion that "if physical signs were present the probabilities were against complete recovery, but if the disease were detected before this there was hope that it might be cured." This statement is as true now as it was twenty years ago. In modern radiography we have a method of diagnosing early cases of pulmonary tuberculosis that Dr. Gee in his day did not possess, it being now possible to discover deep-seated tuberculous disease in the hilus and surrounding pulmonary tissue before tubercle bacilli appear in the sputum and long before physical signs manifest themselves.

Dr. Jordan states that in favourable cases of what he describes as "peri-bronchial phthisis" the X-ray mottling of the lung tissue, indicating active foci of disease, becomes replaced by linear shadows due to the broncho-pneumonic areas cicatrising into fibrous tissue. In progressive cases, on the other hand, the mottling becomes more pronounced

and definite blotches appear due to coalescence of the mottled areas, while in some places the linear shadows may become wider and more clearly defined, due to the accompanying fibrosis. The combination of these two appearances indicates the presence of definite chronic fibroid phthisis, which sooner or later spreads to the apex and superficial portions of the lung. The co-existence of a few crepitations at one apex with well-marked retraction of the affected side of the chest generally indicates advanced fibroid central disease with an apical prolongation—an opinion which may readily be confirmed by an X-ray examination, which will reveal well-marked central blotching and probably "roof-tiling" of the ribs and costal cartilages. A permanent cure sometimes, though rarely, occurs even at this advanced stage, when the whole lung seems to undergo fibrous transformation as in those cases described by the late Sir Andrew Clarke under the term, "non-tuberculous (?) fibroid phthisis." Such cases are usually met with only amongst the better-off classes who are able to secure specially favourable environmental conditions.

Stages of Hilus Tuberculosis.

The stages of pulmonary tuberculosis commencing at the hilus may be classified as follows:

First stage: Pure hilus tuberculosis.—Hilus tuberculosis strictly limited to the tracheo-bronchial glands and the pulmonary tissue of the root area.

Second stage: Peri-hilus tuberculosis.—Hilus tuberculosis spreading some distance outside the root area in the form of linear prolongations, and accompanied by a few scattered foci of tuberculous broncho-pneumonia.

Third stage: Chronic fibroid phthisis.—There is usually extensive central fibrosis and numerous coalescing, and, it may be, caseating patches of broncho-pneumonia, with extensions to the apex or elsewhere, with or without excavation.

The prognosis in the first two stages of hilus tuberculosis is eminently favourable, granted moderate resistive power on the part of the patient and a suitable environment.

In the third stage one can, as a rule, only hope for quiescence and temporary arrest of the disease, though the life of the patient may be greatly prolonged if the disease remains mainly unilateral. The so-called "one-lung cases" belong to this group. Radiography and post-mortem evidence, however, have made it clear that when one lung has reached the stage of advanced chronic fibroid phthisis there are almost invariably hilus changes in the other lung, glandular or otherwise, although the hilus blotches of the apparently sound lung may not in every case be apparent owing to the fact that it may be dragged beyond the middle line and be concealed by the normal median shadow. A consideration of the symptomatic manifestations in conjunction with occasional repeated X-ray examinations will enable the physician to accurately gauge the progress of the disease in these cases.

Treatment.

Little need be said under this head except to emphasise the importance of commencing treatment at the earliest possible stage of the disease. Successful treatment is thus intimately linked up with the question of early diagnosis.

Dr. Gee's aphorism that "therapeutics must begin before physical signs are developed, and if you wait for physical signs you wait too long," exactly sums up the situation.

Professor Karl Pearson's conclusions in respect to the comparative futility of hygienic environmental treatment, as deduced from the after-history of sanatorium patients, are largely fallacious, for the simple reason that fully 90 per cent. of patients admitted to sanatoria are already beyond the curable stage, although many of them are presumed to be in an early stage simply because no definite physical signs can be detected except at one apex, regardless of the probability of extensive underlying central disease.

If a patient suffering from hilus tuberculosis in an early stage is placed under favourable environmental and hygienic conditions, whether at a sanatorium or elsewhere, the active condition as a rule rapidly subsides, and a permanent cure in process of time is effected, so that calcareous nodules and linear cicatrices are left as the innocent witnesses of previous infection.

If, on the other hand, the cases are not taken in hand until physical signs develop, it is generally too late to effect a permanent cure, as an extension to the apex too often means the presence of more or less extensive tuberculous deposit intervening between the root and the surface of the lung.

It is the characteristic X-ray appearances in conjunction with a confirmatory positive tuberculin reaction to which we must trust in deciding when to commence treatment.

It is in these cases of early hilus tuberculosis that carefully administered tuberculin treatment achieves its most signal triumphs. The increasing thereby of the protective properties of the blood and tissues at this stage will serve in most cases to completely check further progress of the disease and pave the way to a permanent cure. This is particularly so in the case of glandular tuberculosis in children. This is the time for strengthening the resistive power of the organism, whether by hygienic methods or by specific tuberculin treatment, or better still by both methods combined.

I would conclude by expressing the belief that the universal recognition by the profession of the importance of hilus disease and its early diagnosis may inaugurate a new era in the treatment of pulmonary tuberculosis.

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